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Diabetes Case Reports

Perspective

Biochemical Effects of Diabetes on the Balance Between Oxidative Stress and Antioxidant Defense System

Chuanlin Shi*

Department of Pharmacy, The Second Affiliated Hospital of Fujian Medical University, Fujian, China.

DESCRIPTION

Diabetes mellitus (DM) is a heterogeneous metabolic disorder characterized by hyperglycemia due to defective insulin secretion, insulin resistance, or both. Type 1 diabetes results in insulin deficiency due to autoimmune-mediated destruction of pancreatic β-cells. Patients need insulin treatment to survive. Type 2 diabetes is characterized by insulin resistance and relative insulin deficiency rather than absolute insulin deficiency. Type 2 diabetes usually occurs in obese people and is associated with hypertension and dyslipidemia. The ability of nutrients to stimulate insulin release from pancreatic β-cells reflects the ability of islet cells to increase the oxidative flux. Oxidative stress associated with insulin resistance and non-insulin-dependent diabetes mellitus also contributes to reduce insulin action. Therefore, treatment is aimed at reducing insulin resistance (diet, exercise, medication) and stimulating insulin secretion.

Defined diabetes

Diabetes is any disorder characterized by excessive urination. The most common form of diabetes is diabetes mellitus, a metabolic disorder that fails to completely remove glucose due to the disruption of insulin function, leading to diabetes and hyperglycemia. Other symptoms of diabetes include polydipsia (excessive thirst), polyuria (excessive urination), polyphagia (excessive hunger), and lipemia. Two key criteria are used to clinically establish a person with diabetes. One has a Fasting Blood Glucose Level (FBG) above 126mg/dl (7mmol/L) and a normal value below 100mg/dl (5.6mmol/L). The second is defined by the results of a glucose tolerance test in which plasma glucose levels above 200mg/dl (11mmol/L) are present at two time points during the Oral Glucose Tolerance Test (OGTT).

Type2 diabetes (DM2): DM2 is a metabolic disorder characterized by hyperglycemia that may be due to defective insulin secretion from pancreatic β-cells, insulin resistance in peripheral tissues, and or excessive accumulation of triglycerides and fatty acid derivatives in skeletal muscle. This condition is a heart, such as complications of micro vascular (retinopathy, nephropathy, neuropathy) and macro vascular (coronary, cerebrovascular, peripheral vascular disease), primarily caused by abnormal activation of physiological pathways. It is one of the main causes of vascular disease. It is also associated with an increased risk of developing cancer, mental illness, cognitive decline, chronic liver disease, and arthritis.

Increased glycolysis

Hyperglycemia appears to improve non-oxidative metabolism (glucose to lactate conversion) by increasing glucose 6-phosphate (G6P). Increased glucose metabolism to lactate is associated with an increased NADH / NAD + ratio. Under these conditions, where glycolysis is significantly accelerated, glyceraldehyde-3-phosphate dehydrogenase is converted to 1,3-biphosphoglycerate (1,3-DPG) of glyceraldehyde-3-phosphate (GAP). Oxidation appears to be the rate-determining step in glycolysis (30), a reaction linked to the reduction of NAD + to NADH. In the cytosol, NADH is oxidized to NAD + by Lactate Dehydrogenase (LDH) and pyruvate is reduced to lactate. Therefore, increasing the NADH/NAD + ratio reflects an increase in the lactic acid/pyruvate ratio.

Mechanism of oxygen free radical production by hyperinsulinemia

Decreased strength, increased body fat percentage, and upper body fat distribution are generally associated with hyperinsulinemia and insulin resistance. Some evidence seems to indicate a link between hyperinsulinemia and free radical production. It was reported that exposure to insulin leads to time- and dose-dependent accumulation of hydrogen peroxidase in suspension medium in intact human adipocytes. This effect associated with the presence of membrane-bound NADPH oxidase was found to persist after cell destruction, indicating that it did not require ATP and bypassed the receptor kinase step. In addition, increased insulin concentration in rats following intraperitoneal injection of dextrose has been found to be associated with increased free radical production.

CONCLUSION

The development of undesirable adverse effects such as fluid retention, weight gain, hepatotoxicity, plasma volume expansion, hemodilution, edema, bone fractures, and congestive heart

Correspondence to: Chuanlin Shi, Department of Pharmacy, The Second Affiliated Hospital of Fujian Medical University, Fujian, China, Email: Shic005@sina.com

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failure, but it is also involved in the prevention of developing atherosclerosis, even though there are certainly another great number of studies which can demonstrate the opposite and also confirm that some agonists of the receptor, specifically rosiglitazone, may increase CVD risk. We believe that the associated risk of Cardiovascular disease (CVD) during TZDs therapy may be related to different transcription patterns in the Peroxisome Proliferator-Activated Receptors (PPARs) activation due to different ligands, since troglitazone and pioglitazone do not increase CVD risk, as pioglitazone may cause bladder cancer, or rosiglitazone or troglitazone or hepatotoxicity, which is directly correlated to the use of troglitazone or the other TZDs, as they interact in different ways with the receptor and therefore induce different conformations and different interactions with coactivators/corepressors, as different interactions with the responsive element, therefore triggering the transcription of diverse genes.